Society Proceedings.

NEW YORK NEUROLOGICAL SOCIETY.

March 3, 1903.

The President, Dr. Pearce Bailey, in the chair.

Tabes Associated with Hemiplegia.—Dr. Joseph Collins presented a man, forty-two years of age, a bar tender by occupation. When twenty-four years old he had had a chancre, for which treatment was given. A year later an iritis developed, and disappeared after two months' treatment. There was nothing further until eight years ago, when he was seized with an attack of vertigo, which was associated with an aphasia lasting several days. There was no paralysis. Four years later there was a similar attack associated with vomiting. Last August the man fell in an attack in which he was only partially unconscious. Examination showed a right-sided hemiplegia, and this had continued ever since. On coming to the hospital, there was found in addition a very marked atrophy of the right shoulder and an absence of both knee-jerks. The pupils were small and regular, and reacted to accommodation but not to light. There were incontinence of urine and manifest ataxia. The case was presented as one of tabes, the latter probably antedating the attack of thrombosis from which the hemiplegia originated. Coincident with the attack of cerebral thrombosis there must have been an obliteration of one of the cornual branches of the anterior spinal artery. In reality, this case presented three conditions: tabes, cerebral thrombosis and a destructive poliomyelitis of very limited extent. Tabes and hemiplegia were rarely associated.

Dr. J. Ramsey Hunt said that he had examined this case in the City Hospital. He had been doubtful about its being an anterior horn disease, and inclined to the opinion that there was a plexus lesion.

Dr. M. G. Schlapp said that in some of these cases of hemiplegia certain groups of muscles were markedly atrophied. The electrical examination should decide whether the case was of central or peripheral origin.

Dr. J. Fraenkel said that in only a few of the cases of tabes with hemiplegia had the knee-jerk returned, and this had only been temporary.

Dr. C. L. Dana said that he had had a short time ago under observation a man with tabes whom he had seen the very day of the occurrence of the stroke. There had not been any return of the knee-jerk, and he thought this could only occur in the early stage.

Dr. Collins said that there had been no suspicion of the existence of tabes before he had examined this man, and he had been deeply interested in the presence of the marked muscular atrophy.

A Case of Major Hysteria (?).—Dr. Collins then presented a woman, forty-four years of age, a cook by occupation. There was no history of alcoholism or of venereal disease. The present trouble began four years ago with an involuntary twitching of the hands and a loss of sensation in the hands and arms. This loss of sensation had gradually spread over the entire body. She had no pain, but felt tired and walked with difficulty. The gait was not strictly ataxic, but was rather shuffling and choreic. There was a peculiar condition of the left lower extremity, the lower third being shrunken without any hardening or thickening of the skin. Examination showed universal analgesia and more or less thermal anesthesia and analgesia. The knee-jerks and ankle jerks were exaggerated. There was slight disturbance of speech but not of intellection. She presented none of the ordinary stigmata of hysteria, and the color fields were of normal size. The woman had not improved under hospital treatment. According to the history, her father and sister were similarly affected, but the former lived to be over seventy years old. The case was presented without a positive diagnosis, because it did not seem to Dr. Collins to correspond to hysteria, syringomyelia or Huntington's chorea.

Dr. Schlapp was of the opinion that the case was one of hysteria, because the condition did not correspond to any known pathological lesion. The condition of the leg might have been brought about by a self-inflicted trauma. Two years ago he had presented to the Society a case of supposed atypical zoster, but subsequent investigation showed that the patient had produced the condition herself by means of carbolic acid.

Dr. R. H. Cunningham said he was reminded of a number of cases that he had had in a family in Richmond, Va., in 1893. Choreic symptoms developed between the ages of fifteen and thirty in the father, two sons and two daughters. One of the daughters had hemianesthesia. He would look upon the case just presented as one of adult chorea.

Dr. Collins said that he had been inclined to look upon the case as one of hysteria, yet this diagnosis presented many difficulties. After having observed the patient carefully for several months he was still more inclined to the diagnosis of major hysteria.

Dr. Pearce Bailey said that he thought the ataxic gait was due to the anesthesia, and that the case was one of major hysteria. With regard to the other case, there seemed to be no doubt that the atrophy of the shoulder was distinct from the tabes. A fall on the point of the shoulder was quite frequently the cause of a paralysis with just such an atrophy.

A Case of Spinal Tumor.—Dr. M. G. Schlapp presented a man, forty-four years of age, with a good previous history. About two years ago a peculiar twitching had been noticed in the muscles over the right shoulder, and about two months later he had begun to suffer pain at the root of the neck and extending into the trapezius muscle. After a time the right arm became weak, and subsequently the left side. This weakness was found to correspond to the muscles supplied by the anterior and posterior thoracic nerves on both sides. There was the Babinski symptom on each side. There were analgesia and diminished temperature sense on the left side. The diagnosis made was an extramedullary tumor involving the fifth and sixth cervical roots of the spinal cord, pressing upon the pyramidal tracts, and on Gowers' tracts, diminishing the pain sense and temperature sense. The tumor lay on the right side, and probably involved the motor roots on the other side. He thought an intramedullary tumor could be excluded. The man was to be operated on.

Dr. Pierce Bailey said he had seen this man last Fall, and at that time there was no sensory disturbance, and the pain dated back two years, but with intermissions. He had formed the opinion then that there was a degenerative lesion rather than a tumor.

A Case of Cerebellar Tumor.—Dr. J. Ramsey Hunt reported a case of cerebellar tumor with degenerations of the posterior columns. The subject of this report was a man of forty-six who had been admitted to Bellevue Hospital in July, 1902, with typical symptoms of cerebellar tumor, although the tumor could not be localized. The cerebellar symptoms had begun three months before, and the patient was under observation for three months. During these six months there was a progressive diminution of the knee-jerks and Achilles jerks on both sides. It was thought to be due to degenerations in the posterior columns of the cord, as had been described in connection with tumors in the posterior fossa of the skull. There was very marked choked disk, and the man's mental condition became one of apathy and somnolence. At the autopsy two small tumors were found, one springing from the dura mater in the median line, and encroaching somewhat upon the right frontal lobe, and the other tumor was in the left cerebellar hemisphere. Both tumors were medium-sized round-cell sarcomata. The spinal cord presented evidence of greatly increased intracranial pressure. Specimens of the spinal cord, both transverse and longitudinal, were prepared, and they apparently showed degenerations of the posterior columns, such as had been described in connection with tumor. The higher levels of the cord were most affected. Lissauer's column was spared, as was the rule. The fibers in Clarke's columns were also usually spared. These degenerations arose from the entrance of the posterior roots into the spinal cord. It was an anatomical fact, that where the posterior roots perforated the dura mater obliquely there was a constriction, and that here the sheath of Schwann was lost. At the same time there was a constriction of the myelin substance, and some of the fibers were deprived entirely of this substance. The speaker then briefly discussed the mechanical and toxic theories, and said that he presented the case as one favoring the mechanical theory.

A Case of Cerebellar Tumor.—Dr. L. Pierce Clark reported the following case: The subject was a man of thirty, who had had both gonorrhea and syphilis, and who used alcohol and tobacco to excess. The present illness began in October, 1901, with headache in the right occipital region. After a time he began to vomit in the mornings, and the right side became weak. Iodide of mercury had been given without benefit. On examination, there was choked disk in the right eye; the sixth nerve was bilaterally weak; the muscles of the right leg were stiff and painful; the head was held stiffly and bent slightly to the right side; all the deep reflexes were exaggerated, particularly on the right side; there was double Babinski; the gait was characteristically cerebellar. A diagnosis was made of a syphilitic tumor in the middle lobe of the cerebellum involving the peduncle. On admission to the Presbyterian Hospital in January, 1903, for operation, an X-ray examination showed the tumor. The man died rather suddenly, and the autopsy disclosed a tumor of the cerebellum. Between it and the fourth ventricle was a large cyst filled with slightly turbid serum. Apparently the growth was a syphilitic gumma that had undergone syphilitic and calcareous degeneration.

A Case of Spindle-Cell Sarcoma of the Cerebral Dura.—Dr. B. Onuf reported this case and presented a specimen. The subject of the report had been first seen on May 2, 1902, and at that time complained of a shooting pain in the left ear and in the teeth, and of attacks of dizziness. There was only slight perception of light; the gait was staggering, there were no marked changes in sensation or in the reflexes; there was typical choked disk. A diagnosis of cerebellar tumor was made. Subsequently the man developed slight paresis of the left facial nerve with fibrillary twitchings of the muscles in this region, and the hearing on the left side became greatly impaired. In walking, the man deviated to the right. He died in December, and at the autopsy a tumor was found at the base of the left cerebellar hemisphere. It was a spindle-cell sarcoma. This could have been enucleated had it been in a more accessible situation.

Dr. Schlapp said that about a year ago he had presented to the New York Pathological Society a case similar to the one presented this evening by Dr. Hunt showing degeneration of the spinal cord. The specimens from his case had been counterstained with acid rubin and the sheath of Schwann was stained. In all of the cases the degeneration extended to this sheath, especially in the cranial nerves, the trigeminus, the glossopharyngeus and the vagus nerves. He had expressed the opinion at that time that the sheath of Schwann might have some protective influence. He did not think the case tended to disprove the toxic theory. There were no sensory symptoms in the beginning, and probably the degeneration was at first confined to the fibers passing from the posterior columns into the gray matter. The poison appeared to be selective in its action, involving only the reflex fibers. The degeneration was particularly marked in the sensory nerves.

Dr. W. M. Leszynsky said that he had been interested in the fact that respiratory failure had preceded cardiac failure in Dr. Clark's case. One of his own patients had died upon the operating table just as the cerebellar abscess was reached. The heart continued to beat for eight or ten minutes after the failure of respiration. If degeneration existed in the posterior columns it must act chiefly on the third or fourth lumbar segment in order to produce loss of knee-jerks; how, then, would one explain those cases in which the knee-jerk was absent for a time, then became well marked, and was alternately present and absent?

Muscle Tonus and Tendon Phenomena; Their Relationship and Interpretation.—Drs. J. Fraenkel and Joseph Collins presented this paper, which was read by Dr. Fraenkel. It was based upon tonometric examinations of 230 patients. The authors stated that at the present day the view most generally held was that the tendon phenomena were expressions of muscle tonus. General clinical experience showed a definite relationship between tendon phenomena and the tonicity of muscles. Hypertonia, next to exaggeration of the tendon jerks, was a symptom of disease of the pyramidal tracts. The tonometer used in this investigation was the invention of the house physician of the Montefiore Hospital. A total of 554 registrations had been made. The 230 cases were distributed as follows: Apparently healthy, 32; cases of tabes, 25; of organic disease of the nervous system, 33; functional disease of the nervous system, 40; pulmonary disease, 70; heart disease, 12, and various other chronic affections, 18 cases. There were 71 registrations classed as hypertonia. It was found to be frequently, although not always, associated with exaggerated Achilles reflexes. Hypotonia was found to be frequently associated with loss or diminution of the tendon phenomena. There was a comparatively small number of neurogene hypotonias in proportion to the cases of general hypotonia. Neurogene hypotonia and absence of diminution of the tendon reflex were found frequently associated. Neurogene hypotonia with exaggeration of the tendon reflexes was present in nine per cent. It was concluded that hypotonia with exaggeration of the tendon reflexes was present in nine per cent. It was concluded that hypotonia produced by damage of the ascending tracts of the spinal cord was accompanied by loss or absence of the tendon phenomena in every instance. In the cases of hypertonia absent or diminished reflexes were present in six per cent of the registrations. In the cerebral group there was found a larger percentage of hypotonias than of hypertonias. In the spinal group 100 per cent gave exaggerated tendon reflex and 95 per cent showed hypertonia. It had been learned that whenever the neurogene tone was markedly increased or decreased the tendon jerks were increased or decreased correspondingly. The authors concluded that disease of the posterior tracts caused hypotonia, and disease of the pyramidal tracts caused hypertonia. There was a large group of cases giving tonicity and normal tendon phenomena.

Dr. G. L. Walton, of Boston, said that in the exhaustive discussion on reflexes and tonus opened by Crocq in 1901, the reader had reviewed the clinical and experimental evidence which tended to show that the reflexes generally corresponded to the tonicity. The exceptions to this rule he explained as illustrations of the fact that the centers for tonicity (at the cortex) and those for the deep reflexes (in the basilar region) were separate. This explanation was not directly discussed, though the limitation of these functions to the regions indicated was seriously questioned. Drs. Fraenkel and Collins had reinforced in a most convincing way the results of Crocq, though their explanation of the exceptional cases differed from his materially. The question was too complicated to be discussed in its entirety, especially in the absence of exact knowledge as to the seat of either the tonus or the reflexes, but such work as had been reported by Drs. Fraenkel and Collins furnished one of the definite steps by which one might gradually mount to a knowledge of this complex subject. We had been so long imbued with the notion that in man, as in the frog, the spinal cord was the center for reflexes and tonus that it was difficult to transfer our study of these functions to the brain. Even in case of disturbed reflex in cerebral disease we were still apt to think of the function of the brain as limited to the withdrawal, or the increase of influence upon the cord, through the pyramidal tract. For a long time the hypotonicity and loss of reflex sometimes found at the onset of cerebral hemorrhage was attributed to the shock conveyed to the cord. At the discussion already alluded to the disputants, while recognizing the importance of the cerebral influence, were by no means in accord with Crocq in limiting the reflex centers and the centers of tonicity to the brain. Dr. Walton said he had several times taken occasion to suggest that, instead of speaking of lower centers controlled, or inhibited, by higher centers, inhibited perhaps in their turn by still higher centers, we should recognize the combined action of all centers, cortical, basal and spinal, allotting to certain regions a predominating reflex function, liable to transference to a lower level on gradual withdrawal of upper level influence. According to this view each reflex movement might be regarded as a resultant of the activity of various loops of different lengths, connected with each other both laterally and vertically. The more he had observed the reflexes in cerebral disease the more he was inclined to accord to the cortex the chief rôle in the deep, as well as the superficial reflexes, and in the tonus. It was difficult, for example, to explain on any other basis the absence of deep and superficial reflexes in the following case: The patient was unconscious and hemiplegic, with a temperature of 105° F., and all reflexes absent up to the time of his death. The Kernig symptom was present, showing, probably, comparative hypertonicity of the hamstrings, but the extremities were otherwise obviously hypotonic. Autopsy showed pneumococcus meningitis of the convexity with encephalitic invasion of the cortex. In other cases of meningitis the reflexes were frequently preserved, but in one case of meningitis coming under his observation, in which both brain and cord were affected, during the stage of rigidity and obvious hypertonicity the reflexes were absent, but reappeared as relaxation set in, first on the side first relaxed. One must evidently look farther than to separation of the centers to explain such conflicting phenomena; in fact, different cases might require different explanations. Possibly it was partly a question of comparative tonicity. In the last case, for example, the hamstrings might have possessed hypertonicity so far in excess of the hypertonicity of the quadriceps femoris as to "snub" the knee-jerk. In long standing infantile cerebral hemiplegia with contracture the deep reflexes were often absent, though hypertonicity was apparent. Doubtless in some of these cases the contracture was in such position as to put the tendons on too great or too little stretch, thus mechanically preventing the reflex. The combination of exaggerated reflexes with hypotonicity was sometimes seen in Erb's syphilis of the cord, especially in the early stages. In a recent conversation with Dr. Courtney he had suggested that the affection about the fibers of the pyramidal tract was sufficient to impede the transmission of voluntary impulse and of tonicity from the cortex, but the reflex stimulus passed the more rapidly, as in neurasthenia, uninhibited by the volitional mechanism. Though not sure that he had thoroughly grasped the explanation of the readers on this point, Dr. Walton felt that they had proceeded along logical lines.

Dr. C. L. Dana thought the authors had established quite clearly the relations of hypertonia to the reflexes, and had given to the neurological world tangible working data. He had not fully understood the explanation of the effect of the brain on hypotonus and hypertonus. Personally, he made use of a working hypothesis which fitted in very well with the explanation given in this paper. In all the sudden insults to the brain occasioned by hemorrhages producing profound hemiplegia there was always absence of the deep reflexes on the paralyzed side, and with it no doubt hypotonia; whereas there was not this loss of reflexes on the non-paralyzed side. After a time this absence gave place to an increase of reflex. If, however, the hemorrhages were more posterior and involved the sensory sphere, there was more definite and more prolonged absence of reflexes.

Dr. B. Onuf referred to a case in which a tumor of the hip and shoulder centers was diagnosed, and the tumor removed, with the result that a very marked hypotonia developed in the paralyzed extremity after the operation, and was present even two months afterward.

Dr. Collins said that Dr. Fraenkel was satisfied with having established the relationship between tonus and reflexes, and they were both particularly gratified with Dr. Dana's statement that in his cases of cerebral hemiplegia he had observed that the farther posteriorly the lesion extended, either into the sensory cortex or the sensory representation of the capsule, so in proportion was the existence of hypertonia or the lateness with which it appeared. This clinical experience fortified very greatly the position taken in the paper.

Dr. Fraenkel said that he too had been delighted at the confirmation of their position by the very large clinical experience of Dr. Dana. If their observations were correct they should be of value in connection with diagnosis of pure lesions of the pyramidal tract associated with hypertonia and of the posterior tract with hypotonia.

PHILADELPHIA NEUROLOGICAL SOCIETY.

March 24, 1903.

The President, Dr. H. A. Hare, in the chair.

A Case of Progressive Bulbar Palsy.—This case was exhibited by Dr. T. H. Weisenberg. The patient had presented herself at the nervous dispensary of the Polyclinic Hospital three weeks previously with the statement that four weeks before coming to the clinic she had suddenly lost the power of speech. This she attributed to the wearing of false teeth. She protrudes the tongue in a straight line and moves it from side to side. There is a fine fibrillar tremor in the tongue. She can not pucker up the lips. There seems to be no involvement of the upper distribution of the seventh nerve. There is no apparent atrophy of the muscles of the tongue or mouth. She has typical bulbar speech. She swallows with difficulty. There is preservation of the pharyngeal reflex. The jaw-jerk is present. The knee-jerk is normal. The gait and station are normal. On laryngoscopic examination, no gross change is observed.

Dr. Charles W. Burr called attention to the fact that it was rare in a case of this kind to find such great involvement of the speech with such slight palsy of the lips as was present in this woman. As a rule in disease of the bulb when the speech becomes so bad as not to be understood there is marked palsy of the muscles of the tongue and lips.

Dr. William G. Spiller said that a curious feature about this case was that the loss of speech came on suddenly. This he thought was an error of observation on the part of the patient, although such a loss may occur suddenly, probably from hemorrhage. This he considered a case of true bulbar paralysis, not a case of pseudo-bulbar paralysis.